

MEDICAL PROCEEDINGS

MEDIESE BYDRAES

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EDITORIAL · REDAKSIONEEL

AUTOMATIC INSURANCE FOR £10,000

FOR SUBSCRIBERS TO 'MEDICAL PROCEEDINGS'

OUTOMATIESE VERSEKERING VIR £10,000

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The details of the various conditions governing this free insurance are published elsewhere in this issue. We wish particularly to draw attention to the condition which requires subscribers to report immediately any incident which may give rise to an investigation.

kriminele aanklag teen die intekenaar kan uitloop;

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Dit dek die intekenaar ook ten opsigte van 'n ondersoek voortspuitende uit 'n voorval wat plaasvind op enige tydstop tussen die tyd dat hy intekenaar vir 1960 word en die einde van hierdie jaar, en die dekking bly van krag ten opsigte van sodanige voorval indien die ondersoek daarna op enige tydstop voor Desember 1962 ingestel word.

Mediese praktisyns sowel as interns kan gebruik maak van hierdie versekeringsbeskerming wat van toepassing op die genoemde persone in die volgende gebiede is:

Die Unie van Suid-Afrika, die Federasie van Rhodesië en Njassaland, Suidwes-Afrika en dit Britse Protektorate Basoetoland, Betsjoeanaland en Swaziland.

Besonderhede in verband met die verskillende voorwaardes wat op hierdie gratis versekeringskema betrekking het, word elders in hierdie uitgawe gepubliseer. Ons wil die aandag veral vestig op die voorwaarde wat van intekenaars vereis dat hulle enige voorval, wat bes moontlik aanleiding tot 'n ondersoek kan gee, onmiddellik moet aanmeld.

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On the further condition that such prosecution or Disciplinary Enquiry shall be in respect of a charge of assault or culpable homicide, or shall be a Disciplinary Enquiry relating to an act or omission on the part of such Subscriber himself;

And provided further that the nature of the Inquest shall be such that the Company in its

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And provided further that the maximum liability of the Company to the Insured shall be the sum of £10,000 (ten thousand pounds) in respect of any single claim.

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4. The Company shall have the right to cancel cover in respect of any insured Subscriber by giving thirty (30) days' notice in writing to the publishers. Upon any such cancellation the Company shall be under no further liability to such Subscriber save in respect of any incident which has already occurred as at the date of such cancellation.

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6. In the event of the Company intimating to the publishers that it will decline liability in respect of any incident which has given or may give rise to a claim under this Policy, then in the event of legal proceedings not having been instituted by the publishers against the Company within three (3) months after the date of such intimation by the Company to the publisher, the Company shall be under no further liability in respect of any costs which may be incurred in relation to any proceeding relating to or arising out of such incident.

PROCEEDINGS OF A CONFERENCE ON CEREBRAL PALSY

SPONSORED BY THE CAPE PROVINCE CEREBRAL PALSY ASSOCIATION

EDITOR OF THE PROCEEDINGS: DR. G. BEINART

Cape Town

In May 1959 about 40 doctors attended a week-end conference on cerebral palsy at the Cape School for Cerebral Palsied Children. This conference was noteworthy in that it consisted of papers and demonstrations by Dr. and Mrs. Bobath whose work in this field has been recognized internationally.

The meeting was sponsored by the Cape Province Cerebral Palsy Association, which had promoted Dr. and Mrs. Bobath's visit to Cape Town, where they conducted a 2½ months' course for therapists on their method of reflex inhibition in the treatment of cerebral palsy.

Among the 40 Cape Town doctors who attended the conference were orthopaedic surgeons, neurologists, neurosurgeons, psychiatrists, paediatricians, specialists in physical medicine, the local maternal and child welfare officer, school medical officers, general practitioners and doctors attached to the Child Guidance Clinic. Several paediatricians and orthopaedic surgeons from Pretoria, Johannesburg, Port Elizabeth, Durban, Kimberley and Bloemfontein also attended. The purpose of this meeting was to familiarize those doctors who work with therapists in the cerebral palsy field, with some of the features of this particular approach to treatment. What follows is a summary of the papers read by Dr. and Mrs. Bobath.

Dr. Bobath opened the conference with a discussion on the clinical aspects and neuropathology of cerebral palsy. Mrs. Bobath then discussed the principles of her method of treatment, demonstrated various types of cases and indicated in her discussion of each child how she would treat it.

On the second day the important question of early recognition of cerebral palsy was discussed and the diagnostic features were well illustrated by slides and pattern films. Mrs. Bobath demonstrated spastic postural patterns in babies and outlined treatment in the very young.

On the afternoon of the second day Dr. Bobath read a paper on orthopaedic measures in the neurological treatment of cerebral palsy and Mrs. Bobath discussed the link-up of her treatment with the work of other therapists—the occupational therapist, speech therapist, and the follow-through at home.

All papers were followed by discussion and the morning of the third day was devoted to a round table conference.

A suspension type chair, which has been found useful at the Cape School for Cerebral Palsied Children, for cases which have not yet achieved sitting balance was demonstrated.

A SUSPENSION TYPE CHAIR FOR SEATING CEREBRAL PALSID CHILDREN

The seating of the cerebral palsied child constitutes a difficult mechanical problem, since the same seating design may have to support the child with spastic and atonic musculature and at the same time accommodate the athetoid child without trauma

or restriction. The so-called relaxation chair in current use is, in fact, a badly fitting splint which has to be designed to the individual requirements of the child.

The Coplans chair, which was designed at the Cape School for Cerebral Palsied Children and which has been used there successfully since 1954, is based upon the principle of controlled suspension of the child (Fig. 1). The child is fitted into a light canvas corset of the breeches buoy type which extends as high as the axillae and offers attachment to 4 helical springs by 2 anterior pectoral rings and 2 posterior scapular rings. The tension of the springs may be so adjusted that the child is able to sit on a chair without back or sides, the suspension apparatus giving the necessary support and control and exercising at the same time an educative balancing effect. The child may be suspended from a 'halo' type adjustable frame or by a simple 'parachute' type suspension. The chair will accommodate successfully children who suffer from spinal, pelvic and hip joint deformities.

DR. BOBATH'S OPENING ADDRESS

In his opening remarks Dr. Bobath mentioned that he would not approach the problem in a formal and systematic manner, but would tell the conference something of their special approach and highlight certain problems which were still matters of controversy. In cerebral palsy there were hardly any fields in which there was not a great deal of confusion and discussion. For instance, a satisfactory and generally acceptable definition had not yet been arrived at. In England it was felt that cerebral palsy comprised a group of conditions caused by maldevelopment of or damage to the brain of a non-progressive character, which led to a dysfunction of motor action.

The term non-progressive is to some extent already misleading because what is non-progressive is the lesion, whereas one could say that the symptoms, as such, are to some extent progressive, in that they unfold in the babe, develop as the infant grows, and only later achieve a stationary character. We exclude conditions of an hereditary or progressive character, for example the various cerebellar ataxias, Friedreich's ataxia, Krabbe sclerosis and phenylketonuria.

* Designed by the principal of the School, Mr. D. D. Martin.

Dr. Bobath felt that there was a great difference between symptoms produced by cerebral palsy and those produced by conditions of an hereditary and constitutional character. Cerebral palsy usually causes damage of a non-systemic character (it does not pick out certain systems as do cerebellar or Friedreich's ataxia but it cuts across, producing lesions of a mixed type as one might expect from the nature of the causative factors such as prematurity, anoxia, rhesus incompatibilities and haemorrhage).

The definition is really not complete because it leaves out the sensory side and the many associated sensory conditions which have to be taken into account, not only from the point of view of the clarity and completeness of the definition, but also from the point of view of treatment and assessment of the associated mental handicap. Sensory disturbances are frequent and varied and include visual, hearing, speech and proprioceptive disabilities.

Among visual defects (which probably occur in approximately 60% of cases) are found varying degrees of squint, both fixed and alternating; lack of dissociation of eye and head movement, partial or total field defects, and blind-

ness associated with optic atrophy or, more often, visual agnosia due to cerebral damage.

Hearing defects include partial or pitch deafness, commonly associated with the sequelae of kernicterus; also total auditory imperception or auditory agnosia, in which there is inability to recognize and interpret what is heard.

Pure cases of sensory aphasia are probably rare. They are usually combined with a movement defect of the speech organs with spasticity, athetosis or ataxia. Peripheral pseudobulbar palsy also occurs.

Among other sensory disturbances, the most important are the agnosias affecting the whole body or part of it. In these there is a disturbance of building up of body image due, possibly, to the lack of experience of the severely handicapped child who is unable to touch and feel his own body, handle objects and put things to his mouth.

Dr. Bobath stressed that he and Mrs. Bobath believed that every case had to be looked on as a sensori-motor disorder. The proprioceptive system which in infancy served to develop our body sense may either be damaged or, though undamaged, it may be unable to serve its function while the child is able to move only in

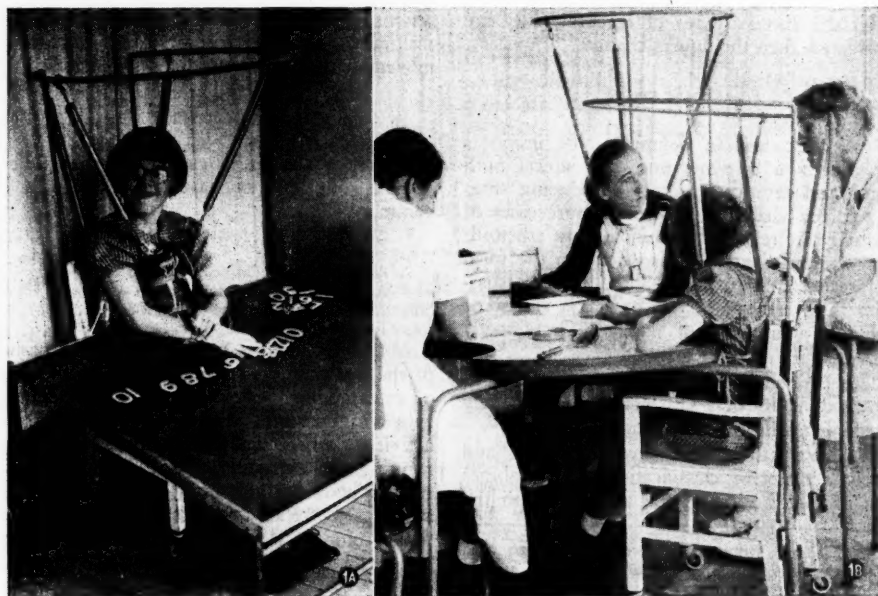


Fig. 1A. A spastic child sitting erect in a suspension chair. The child is using magnetic letters on a metal table.

Fig. 1B. Two suspension chairs. The girl in the foreground has deformities of both hip joints and can only sit satisfactorily in suspension.

an abnormal manner and with undue effort. Treatment is always considered as a sensori-motor re-education and must be integrated with a total approach to the child.

Since Phelps made his first survey at Baltimore in 1943, in which he believed the incidence of the condition was 7 per 100,000 of population, things have changed considerably. In various successive counts in England in various country towns, it has been found that the incidence appears to be very much higher. We now believe that the incidence is nearer 2 per 1,000 of population between 5 and 15 years. It is important to realize this, because it means much greater provision has to be made for the treatment of cerebral palsy than was originally planned.

With regard to aetiology, Dr. Bobath said he would not discuss it in detail. He indicated that approximately 60% of all cases are caused by factors operative in the perinatal period, such as anoxia and haemorrhage; about 30% are caused by factors *in utero*, and belong probably to the group of disturbances at the conceptual period of foetus, such as maternal infections including rubella. Only about 10% are caused by damage which takes place after birth. This is speaking of the condition as a whole, for if the specific types of cerebral palsy are considered, then the figures change.

CLASSIFICATION

As was discussed in definition, the lesion is not usually a systemic one, but affects both motor and sensory structures producing cases of a mixed character. Whereas pure cases of spasticity are relatively common, most athetoids show various degrees of spasticity in the form of intermittent spasms.* Some cases of spasticity show either athetosis or ataxia and some athetoids a mixture of ataxia. Occasionally cases of pure ataxia are found.

This is one of the main reasons for the well-known difficulty of a satisfactory and generally accepted classification of cases.

We have used the following simple and purely descriptive classification. It combines type of distribution of the affection with the

nature of the abnormal muscle tone in a purely descriptive manner:

1. *Diplegia*: The whole body is affected, the legs worse than the arms.

2. *Quadriplegia*: The whole body is affected, but the arms as much as the legs, or more so than the legs. Due to the influence of the asymmetrical tonic neck reflex being more active on one side than on the other, there is usually a more or less well-marked asymmetry of distribution. This type of quadriplegia has also been called double hemiplegia.

While spastic conditions may occur both among the diplegias and quadriplegias, athetosis is more frequent among the quadriplegias.

3. *Hemiplegias*: Most of these are spastics, though some may show, besides, athetoid movements of the distal parts, hands and fingers or toes.

4. *Paraplegias*: In our experience they are rare, most of them being diplegias in whom the arms are only slightly affected.

5. *Monoplegias*: They also are rare, as most of them are hemiplegics in whom the arm is so slightly affected as to escape notice. Both among the quadriplegias and diplegias we find occasionally cases of spasticity or athetosis with elements of ataxia.

In infants a state of 'cerebral flaccidity' is frequently seen. These babies may develop either spasticity or athetosis later on.

The term 'rigidity' in cerebral palsy is difficult to define. In some cases it is only a state of severe spasticity approximating decerebrate rigidity. In other cases it may be due to lesions of the basal ganglia, though some cases may also show the rigidity and paucity of movement known in frontal lobe lesions.

THE MOTOR HANDICAP

In spite of the great variety of the clinical picture, all children have these features in common:

1. An absence of development of normal movement patterns;

2. An abnormal quality of muscle tone;

3. A disorder of the coordination of muscle action both for the maintenance of posture and the performance of movements.

In order, therefore, to understand the nature of the handicap we have to define the function of muscle tone. Sherrington has defined this as serving posture. During every one of our activities, when sitting, standing, walking or running, we constantly change the relationship of our body and its point of gravity to the support. If we are to maintain our equilibrium while moving, we have to adjust our posture to constantly changing conditions. For this purpose the central nervous system brings about a constant shift and redistribution of tone throughout the musculature of the body. This is done automatically and without our conscious participation. A group of postural reflexes, or rather reactions, which is predomi-

* Apparently synonymous terms which are used to denote a combination of athetosis and spasms resembling spasticity are:

Athetosis with intermittent tonic spasms (Bobath);
Tension athetosis (Phelps);
Alternating rigidity (Collis);
Cerebral dystonia (Polani).

In these cases the muscle tone is unsteady and variable. (*Editor's note*).

nantly sub-cortically integrated, serves this function. To them belong the righting reactions of Magnus, and the equilibrium reactions of Weisz, Rademaker and André-Thomas.

These reactions are typical and common. They have arisen during Man's phylogenetic development and have been retained as useful and serviceable. They are responsible for the similarity of man's fundamental behaviour; for the fact that we react similarly when in danger of falling, when we sit, get up, stand and walk.

At birth these reactions are only very inadequately developed. They appear in the growing organism in a definite sequence, in step with the maturation of the central nervous system. These postural reactions form the background against which all our daily activities take place. Without their full and unimpeded development, the normal performance of higher skills becomes impossible.

THE DEVELOPMENT OF OUR MOTOR ABILITIES

We may perhaps say that from birth onwards the newborn develops his motor ability in two directions:

i. With the development of the postural reactions the baby gains the ability to raise his head in prone and supine positions, to turn over from supine to side-lying and prone, to sit up, crawl and stand. This is gradually achieved with the development of the more primitive righting reactions. Later on, from the sixth month onwards, the gradually developing equilibrium reactions give him the ability to preserve and adjust his balance in sitting, standing and walking.

The righting reflexes come into play from birth onwards, reach their maximum effect around the 10-12th month, and gradually disappear around the end of the fifth year. They guide the baby throughout his quadrupedal stage. They are then modified and probably built into the higher integrated and more complex equilibrium reactions.

To them belong:

1. *The Neck Righting Reflex Acting on the Body.* This is present at birth. Turning of the head to one side is followed by a rotation of the body *in toto* to the side, following in the direction of the head.

2. *The Labyrinthine Righting Reaction Acting on the Head.* This reaction comes into play around the 6th-8th week. At first it is weak and the baby raises his head in prone. Later it gains in strength and around the 6th month the baby raises the head in supine.

3 and 4. *The Body Righting Reactions Acting on the Head and the Body Righting Acting on the Body.*

These appear around the 8th month, and serve to secure the normal position of the head in space (together with the labyrinthine righting reaction) and to assure the proper alignment of head, neck, body and limbs.

5. *The Optical Righting Reaction.* This is an important reaction, gaining in importance as a child grows. In the adult the maintenance of the normal position of head and body by vision is, of course, the main factor, compared with which the other senses are of secondary importance. In the young, however, the optical righting reactions only attain their main function once the normal position of the head in space has been attained and secured with the help of the other righting reactions.

The equilibrium reactions, like the righting reactions, appear in a definite sequence. They are very complex and require for their proper function probably the interplay of basal ganglia, subthalamic nuclei, the cerebellum and the cerebral cortex. They maintain equilibrium of the body, maintain and adjust our posture and remain active throughout life.

ii. As mentioned before, the abilities of the newborn develop also in another direction. Gradually, as the brain matures, the higher centres come into play, the primitive total reflex patterns of the newborn are broken up and elaborated in various ways. Out of the total grasp reflex of the newborn develops the ability of opposing the thumb and picking up a small object with thumb and forefinger. This is achieved around the 15th month. This development is characterized by the increasing ability of inhibition, the ability to suppress more and more of any unwanted activity and to restrict the movements to smaller segments of the body.

NATURE OF MOTOR HANDICAP IN CEREBRAL PALSY

In the cerebral palsied child there is an interference with the developmental process. We find, as a result of the lesion, a lack of sufficient inhibitory control and the release of the tonic or static postural reflexes described by Magnus. Therefore in all children with cerebral palsy the preservation of the total reflex character of their movements is characteristic. Furthermore, we find tonic reflexes which produce spasticity or intermittent spasms, although it may be wiser to say that the tonic reflexes are co-existent with spasticity or intermittent tonic spasms.

These reflexes dominate the motor behaviour of the child and fix him either permanently or intermittently in abnormal positions. They either prevent or distort the intended movement. The child can therefore either not move at all, or only move within the patterns of

these abnormal reflexes. In their pure form, these patterns are only seen in the very young or in some very severe case. In the milder cases they are usually modified by the child's effort to make some use of these patterns for purposive activity and to compensate with the less affected parts for the shortcomings of the severely involved parts. These efforts may in time produce typical deformities and contractures.

The most important tonic reactions are the asymmetrical and symmetrical tonic neck reflexes, the tonic labyrinthine reflex, the positive supporting reactions and the associated reactions.

1. *The Asymmetrical Tonic Neck Reflex.* Turning of the head to one side produces an increase of extensor tone in the limbs to which the face is turned, and an increase of flexor tone in the opposite side (*Fechterstellung* of the Germans.)

In its clear-cut form, as in more severe cases, the face limbs extend, the skull limbs flex. However, the reflex effect is usually more marked on the arm than on the leg. In some milder cases the reflex effect can only be detected by testing the limb for resistance to flexion or extension with the head turned towards or away from it.

Frequently the reflex acts on the eyes in such a way as to fix them. If the head is turned to the right the eyes are fixed at some point to the right of the midline and cannot be moved to the left beyond the midline. Thus the child is prevented from looking at an object and grasping it while looking at it, and has to turn his head away in order to close the fist and flex the arm. He cannot move the object to his mouth but only to the back of his head. He is unable, therefore, to acquire hand-eye co-ordination, to bring his hands together in midline, to mouth objects or to touch parts of his body with his hands.

This reflex is usually most marked in the quadriplegias, when it is stronger on one side, usually the right. The child learns to use the flexed left hand for some manipulative activity. His head is usually held to the right. In time he may develop a kyphoscoliosis, flexor deformities of the hips and knees through prolonged sitting, and is in danger of a subluxation of the left hip joint.

2. *The Symmetrical Tonic Neck Reflex.* This reflex is produced by raising or lowering the head. Raising the head produces an increase of extensor tone in the arms and an increase of flexor tone in the legs; lowering of the head has the opposite effect.

The child in kneel-sitting with the head raised will show extended arms and the legs may be locked in flexion. He is therefore unable to go forward on his knees to four-foot kneeling, and is unable to crawl. As the child lowers the head, the arm flexes, the legs extend and, if this is marked, he may shoot forward on to his face.

In the diplegic child the reflex may only be seen in its effects on the legs. The child seems to be fairly normal in kneel-sitting, but can only move by hopping forward without extending his hips. If the reflex is less strong, the child may just be able to move forward to four-foot kneeling, but will crawl with very poor alternating extension of the hips. In turning around, he pivots the body around the flexed knees.

3. *The Tonic Labyrinthine Reflex.* The otolithic organ of the labyrinth produces in the cerebral palsied child maximal extensor spasticity in the body and limbs in the supine position, and minimal extensor spasticity with a relative increase of flexor spasticity in the prone position. Intermediate degrees of extensor spasticity are produced by intermediate positions between these two extremes. Decisive for the result is the position of the head, and with it the otolithic organ, in space.

In the more severe cases the extensor spasticity in the supine position is very distinct. The head and neck are retracted, the shoulders pulled back, the spine arched, the legs extended, adducted and rotated inwards, sometimes crossed. The child is unable to raise the head and to sit up. The action of the labyrinthine righting reflex in raising the head is therefore prevented by the severe extensor spasticity. The child cannot bring his arms forward, cannot bring them together in midline, nor can he use his arms to pull himself up to sitting. If he turns his head to the side, he will not turn the body over to the side, as the action of the neck-righting and body on body-righting reaction is prevented by the severe retraction of the shoulders. He may instead show an asymmetrical tonic neck reflex.

In prone he usually shows strong flexor spasticity. The head is on the support, either turned to the side or with the face down. The shoulders are flexed forward with one or the other caught under the body. The spine is flexed. The hips are flexed, the legs usually extended. The head cannot be actively raised as the action of the labyrinthine righting reflex is inhibited by flexor spasticity. The child cannot extend his arms to support the body weight as there is inhibition of the protective exten-

sion of the arms by the flexor spasticity of the arms, i.e. inhibition of *sprungbereitschaft* of Schaltenbrandt. He is unable to get up kneeling, etc.

In sitting the child learns to use the head position to strike a compromise between flexor and extensor spasticity. He sits on a narrow sitting base, often far back, due to insufficient flexion of the hip, and brings his body over to his point of gravity by flexing his dorsal spine. The neck is shortened, the chin poked forward. If he is made to look up, he raises the head, moves into a position of increased extensor spasticity and falls backwards. If he is made to lower the head, he falls forward, pulled forward by increasing flexor spasticity. If he has besides an asymmetrical tonic neck reflex, he may show a scoliosis as well as a kyphosis.

In young children with severe extensor spasticity the Moro reflex may remain active well beyond the sixth month. It can be seen in supine and in sitting, when if the child startles and looks up he is in danger of falling backwards.

4. *Associated Reactions—so-called by Walshe.* Associated reactions are produced by overflow of stimulation. The overflow is either produced by effort by a sound limb on to the affected limbs, or the effect may be from one affected part on to the other. The result of the effort is an increase of spasticity of the affected parts with an accentuation of the abnormal posture. It is easily demonstrated on the hemiplegic patient in whom an increase of the flexor spasticity of the hemiplegic arm can be demonstrated by asking him to squeeze an object with the healthy hand or to stand on the sound or spastic leg. The degree of overflow is in direct relation to the amount of effort.

The practical importance of these reactions is that one cannot in a spastic child afford to treat any one part of the body. Attention in treatment to, say, the spastic arm or hand only, may improve the hand or arm, but may worsen the condition of the other parts.

5. *The Positive Supporting Reaction.* This is produced in the standing patient by the two-fold stimulus of touch of the ball of the foot on the ground, and pressure with stretch of the intrinsic muscles of the foot. The result is a stiffening of the leg, with increase of both extensor and flexor spasticity (so-called co-contraction). The leg becomes a stiffly extended pillar. In the spastic child who walks on tip-toes, every step brings the ball of the toes in contact with the ground, with subsequent pressure on it. The result is that at this stage of every step, spasticity in the

leg greatly increases and may throw the patient backwards. The difficulty of walking is considerably aggravated by the action of the crossed extension reflex, which further increases the spasticity of the standing leg, when in walking the other leg is flexed.

These are the main tonic reflexes, active either in a permanent or in an intermittent manner in the cerebral palsied child, and their combined actions explain well the nature of his motor difficulties.

TREATMENT

In treatment we aim first at the inhibition of these abnormal reflexes. Once their influence is inhibited, and muscle tone more normal, we can start to lay down normal movement patterns on a normally functioning proprioceptive system. We can then facilitate the normal righting and equilibrium reactions in their proper developmental sequence. We call this process facilitation, as experience has shown us that the patterns of these reactions are potentially present and that they do not therefore have to be taught. They often make their appearance almost spontaneously, once the tonic reactions, spasticity or intermittent spasms, have been successfully inhibited.

PRINCIPLES OF TREATMENT

Mrs. Bobath, gave a most interesting paper in which she analysed the faulty movement patterns in cerebral palsy, and discussed her approach to treatment. She reiterated the 3 main factors which interact with one another, and which, she felt, were responsible for the cerebral palsied child's motor disabilities:

1. An absence of normal development of movement patterns;
2. The abnormal quality of muscle tone;
3. The abnormal coordination of muscles in posture and movement, due to released tonic reflex activity.

The Bobath treatment was based on neurophysiological lines, because they felt that cerebral palsy was a neurological and not an orthopaedic problem. The damage to the brain led to a disorder of coordination of muscle action and not to paralysis or paresis of individual muscles. The whole of the patient was affected and therefore it was not adequate to treat any particular muscle group or joint at a time, or an arm or leg, a hand or foot one after another. Treatment was quite unlike that of a patient with a normal central nervous system who could control his muscle functions and who if asked, for example, to move his

elbow would do so if his muscle power permitted it. The cerebral palsied patient had all the muscle power he needed and his nerve supply was unimpaired. But he was unable to direct the impulses to his muscles in the normal way. He could not contract or relax individual groups of muscles or move parts of his body independently of others. He had abnormal coordination in posture and movement throughout his body.

The muscles of the normal and of the spastic patient are coordinated in patterns for every movement. Isolated muscles are never used for any movement. The musculature of the whole body is activated in ever-changing patterns for every movement that is performed. A movement of one part of the body calls for synergic action of other muscle groups, for smooth and regulated activity of the antagonists and, most of all, for a constantly changing postural adjustment of the rest of the body to changes of equilibrium. These changes of muscle tone and posture which accompany a movement are sub-cortically integrated and controlled. We think of walking or grasping an object, but we do not think of the muscles we use. We initiate the action, but the details of its execution are controlled sub-cortically, i.e. a large part of every voluntary movement is automatic and outside our consciousness.

The cerebral palsied patient's postures and movements are also coordinated in patterns, but in abnormal patterns. These are not as varied as in the normal. The patient has only a few stereotyped patterns of motor activity at his disposal. These patterns are widespread and involve large parts of the body or even the whole body. They do not allow for small parts of the body to be moved independently of others, and therefore they are totally inadequate for fine skilled movements.

The postural patterns of the child vary in different positions, but with minor individual differences they are similar in supine, prone, sitting, kneeling and standing in all children with cerebral palsy. The distribution of spasticity through the muscles of the whole body changes in a predictable way with every change of position. Spastic resistance of the muscles, which fix the patient in these postures, prevents movement. These abnormal patterns are due, not only to released tonic reflex activity, but also to voluntary compensatory activity whereby the patient tries with the less affected parts of his body to compensate for the inadequate and abnormal reflex patterns of the more affected parts. He also uses reflex synergies in order to sit or stand or use his hands. Except

in very severe spastic quadriplegics who cannot move and show only a few tonic reflex patterns, one sees a mixture of abnormal reflex and compensatory voluntary activities.

Mrs. Bobath went on to say:

'Our aim is to give the child control over his abnormal patterns of posture and movement, and to help him to change them to more normal ones. This means not only gaining more normal and more individualized movement patterns, but reducing spasticity or involuntary movements throughout the whole body. As a first step we try to gain control over and then help the patient to inhibit the tonic reflexes which are responsible for the typical abnormal postures. We have found that we can suppress, or inhibit, these overactive tonic reflex activities by placing the child in certain new positions which are the extreme opposite of his former postures. We call these new positions "reflex inhibiting postures."

When the patient is adjusted to the reflex-inhibiting posture, there is no longer resistance from spastic muscles, and involuntary movements are absent; muscle tone is normal for the time being, and we can gradually release our hold. As we do this, we slowly hand over control of the new posture to the child, who then starts holding it actively, first with some guidance, later on alone. In this way the child learns to control his abnormal postural reactions, i.e. he learns to stop them. But he also learns to use his muscles in new patterns of coordination for holding and moving in the new postures, or from one to another.

The moment muscle tone is normal, or near normal in reflex-inhibiting posture, we can start moving the child. Our first aim is to get normal active movement responses to our handling. We call this facilitation of movements. For instance, we may turn the child's head and he will roll over to his side in a normal way. We may then lift his head high up while turning him further around, and he rolls over to his abdomen and may put his hands down to support himself. In this and many other ways the child learns and feels the patterns of the normal fundamental movements. For this facilitation we use righting reactions, i.e. we make the body follow movements of the head, or we move the legs and make the body follow, we also might move the trunk and make the head right itself in space. At the same time we stimulate balance reactions in various activities. We build these movements up through all stages of their development in normal children, from lying

and turning over, to sitting up, to kneeling and getting balance in sitting and kneeling and crawling. The child learns to use his hands for support and holding on, he learns to stand and walk. When we move him, he learns to right his body, and when we disturb his balance, he learns to get it back automatically. Later he will use these automatic movement patterns for voluntary movements and for functional skills. He will have sufficient balance in sitting to use his hands freely, he will be safe enough on his hands and knees to crawl, and he will be able to transfer his body-weight from one leg to the other without losing balance, so that he can start to walk.

From all this you will see that we prepare the child for every movement before we expect him to do it in a fairly normal way. We analyse his difficulties, we find out why he cannot do a certain movement, or why he does it abnormally, what interferes with it. Then, instead of teaching the movement, we first inhibit the abnormal activities which prevent it.

In the young infant and child, who has not yet developed strong spasticity or athetoid movements, treatment leads to quicker and better results, because there are not so many abnormal reactions to inhibit, and there are hardly ever contractures and deformities.

The treatment of the older child, who has already strong spasticity or athetosis, takes longer and is more difficult. Results depend very much on the strength of abnormal reflex reactions, and on how far the child can learn to control them. They depend also on the presence and severity of contractures.

Our whole treatment is directed towards making the child *feel* a normal posture, a normal movement. Cerebral palsy is a sensorimotor disorder. The child with cerebral palsy experiences only the sensations of his abnormal muscle tone, of his few and abnormal postures and of very limited and incorrect movement patterns.

The teaching of skills by making the patient use the abnormal patterns of coordination to the best advantage has its dangers, even if he learns to feed and dress himself, to write, stand and walk. The repetition of abnormal performance often leads to deformities which are difficult, and sometimes impossible, to correct afterwards. Whatever skill he may learn, whether it be walking, feeding, dressing, etc. will be based on and guided by these abnormal sensations. Therefore any newly acquired activity will only perpetuate and reinforce the abnormal sensori-motor patterns. There is also

the danger of deformities, especially in spastics with so few patterns.

In treatment, therefore, we must give the child as many normal sensory impressions of movement as possible, so that he can start laying down new and normal sensory patterns of posture and movement, and build up new activities based on these normal kinaesthetic images.

THE EARLY RECOGNITION OF CEREBRAL PALSY

It was soon noted that the results of treatment could be greatly improved if treatment could be started as early as possible, i.e. at least before the end of the first year. The effects of the brain lesion are not usually apparent during the first few months of life. Tonic reflex activity as a waning and insignificant influence in the behaviour of the newborn up to the end of the 4-6th month, is physiological. This is seen, e.g. in the tonic neck reflex. Depending on the severity of the case, the earlier tonic reflex activity becomes stronger, usually, first in an intermittent form, and later spasticity increases and the picture becomes clearer. This may happen quite early, around the 4-6th month in severe cases and these cases are not usually in doubt.

In the milder cases, in whom either only part of the body is affected, e.g. the hemiplegic child or the predominantly paraplegic child, symptoms of brain damage are not usually recognized until later, rarely before the end of the first year. Many athetoids will not show involuntary movements before the end of 18 months to 2 years.

If we could make a diagnosis before the onset of definite symptoms and start treatment, we might hope to get better results for the following reasons:

1. We might allay the worsening of the condition and prevent the development of severe spasticity and other abnormal signs.
2. Handling is easier, inhibition much more easily obtained, and facilitation in the developmental sequence much more successful and promising.
3. No abnormal habits have to be destroyed, contractures and deformities be prevented and surgical procedures may subsequently become quite unnecessary.

Furthermore, as McKissock has pointed out, the young and immature brain has powers of adjustment and compensation not usually available to the more mature brain. We may therefore hope to achieve better results with early treatment, than at any other time later on.

But perhaps the most important reason is that by early treatment we can hope to prevent,

or at least allay, a possible mental retardation resulting from the physical handicap. This applies especially to the more severe case who, as a result of the physical handicap, cannot gain the necessary experience by exploring his body and the environment and who may therefore develop a secondary mental retardation on top of the motor handicap. One must keep in mind that the first 2 years of the child's life is the period of tremendously fast development, and that it is rarely possible to make up for lost time if much of this time has been allowed to lapse without helping the child.

I do not think that we have so far solved the problem of assessing with any accuracy the mental endowment of a child of under 18 months or even older, suffering from cerebral palsy. After all, we assess the mentality of these children more or less by measuring their physical development, assuming that this is an expression of mental development.

What then are the difficulties of an early diagnosis? To some extent they arise out of the first-mentioned factor, viz. that the influence of abnormal reflex activity is not evident at first. Indeed, you may believe that the tonic reflexes are a normal phase of the newborn baby up to the end of at least the 4th month. I cannot quite agree with this view. We believe that even in babies under 4 months tonic reflexes, as e.g. the asymmetrical tonic neck reflex reactions, are present only in a very mild, waning form. The child may assume the tonic neck reflex attitude at rest for a little while, but never in his development is the baby's behaviour dictated by any of the tonic reflexes. They make their influence felt only in certain fleeting or temporary attitudes, from which the baby can easily move away. It can be argued, of course, that in the child with cerebral palsy these reflexes are exaggerated and not strictly pathological. However, the fact remains that any sign of exaggerated activity of these tonic reflexes is a sign of abnormality and, in our opinion, the certain sign of brain damage, i.e. of cerebral palsy.

Parents usually bring their children to the doctor when they notice that he fails to achieve any of the essential landmarks, so well known from the studies of Gesell. This is especially true in the case of babies with a stormy and abnormal birth history. The task before the doctor is then the differential diagnosis of general motor and/or mental retardation, retardation due to other diseases, such as the myopathies among others, and retardation to cerebral palsy.

Diagnosis, as always in medicine, must be based on a thorough history and case-taking, a general medical examination, and specific neurological and paediatric assessment. The baby often falls between two chairs: paediatrician and neurologist. The conventional examination is not very helpful, and is very difficult. The examination presupposes a good working knowledge of the motor, mental and emotional behaviour of the normal baby and a more thorough than usual knowledge of the changes of behaviour at various stages of development.

In our examination of the baby we seek signs of abnormal reflex activity. We seek to explain the retardation as caused at this stage, i.e. at the time of examination, by the still insidious influence of the tonic reflexes. We use this method of examination in conjunction with all the other established methods of examination.

Before I go into a description of these special tests, let me first mention a few controversial points of diagnosis arising out of the interpretation of the more usual and customary tests of examination.

i. André-Thomas and Peiper state that in the normal baby the Babinski sign or plantar extensor response is characterized rather more by its fickleness, its unpredictability, than otherwise.

They believe, and I am inclined to agree, that a permanent and always recurring plantar extensor response is very suspicious of brain damage.

ii. The eye coordination of the baby is poor, fusion of the eyes developing slowly. A permanent internal squint is, in our opinion, highly suggestive of brain damage.

In doubtful cases, Dr. Bobath advised that babies be referred back in 1 week—not in several months' time. Where the diagnosis was in doubt, Dr. and Mrs. Bobath advocated treatment.

Dr. Bobath then showed numerous slides and films to illustrate the diagnostic points in various positions. He made special mention of the 'frog' or 'floppy' child where primitive patterns of movements and delay in achieving head control are points to consider, in the early diagnosis of the athetoid infant.

Among the points illustrated were the following:

In the Normal Infant

At birth and up to 4 weeks: The normal baby usually shows some ability to support its head on the 'pull-to-sit' manoeuvre.

Prone, the head was turned to one side, although the general attitude was one of flexion with hips, knees and ankles flexed. The arms were usually at the infant's sides.

4-6 weeks: Head raised in prone.

6 months: Head raised supine. Established extension of arms. Puppy position on forearms with extension of neck and spine.

In a normal baby considerable stimulation is necessary before the Moro reflex is elicited. In cerebral palsied baby the Moro reflex is far more easily elicited.

8-10 months:

(a) Body-righting reflex on body.

(b) Body-righting reflex on head.

(c) Modified neck-righting reflex and a spiral reflex is introduced between shoulders and trunk. This introduces rotation as a means of getting up from supine.

In the Affected Infant:

1. *Supine*: Maximal extensor spasticity in supine.

2. *Poor head control* on 'pull-to-sit'.

3. *Prone*: Position of minimal extensor spasticity in which infant lies on its airways with trunk and legs extended.

The infant may raise its head by total extension. Extension is weakened by flexing hips, e.g. and then the infant is unable to raise the head. Pulling up of arms and poor head control may be the only signs of flexor spasticity.

4. *Landau reflex*.

5. *Absent protective extension of the arms* when the infant is held by its trunk in the prone position.

6. In holding the infant erect, note pointing down of the feet. Tipping up and down accentuates pointing of the toes and produces typical scissoring with adduction and internal rotation of legs.

Mrs. Bobath then demonstrated the treatment of some very young children which, she stated, differed from that of older children in some respects.

Firstly we do not yet have to counteract existing deformities. We know what type of deformities they might develop and therefore we have to anticipate and prevent such developments.

Secondly, spasticity is usually not yet very strong, and athetoid movements do not occur before the 18th month or even the 3rd or 4th year. In the young athetoid we often find a state of apparent 'flaccidity,' alternating with intermittent spasms. It seems to us that athetoid movements occur only when the child tries to do voluntary movements, or to get up

and maintain the upright position in sitting and standing. Therefore we first give the athetoid child a steady background of muscle tone, one that does not fluctuate between too high and too low. Then, when he tries to move voluntarily, he will have more normal postural control of head and trunk and this will make the movement of his limbs more controlled. Spasticity in young children can be reduced much more quickly and easily than later on, by moving the child a great deal, using the early and primitive movement patterns first, but at the same time keeping him out of his abnormal postural patterns.

Thirdly the young child has not yet established abnormal sensori-motor patterns. He has not yet tried to do things in an abnormal way. Because of this, we can help him to get normal sensations of movement. We make the child go through the various stages of normal motor development, i.e. through all stages which prepare for and lead up to walking and the independent use of the hands. We get control of head and trunk in lying prone, supine, in sitting, kneeling and standing. We get him to turn over, to sit up and to get on his hands and knees and to crawl and later to stand up. We help him to use his arms and hands for support in extension, e.g. in kneeling, crawling and sitting and in getting up to his feet from kneeling. During all these activities we develop his balance reactions. Tonic reflex activity and intermittent spasms have to be inhibited by avoiding positions in which they are strongest. But at this early age they are usually not very strong and the facilitation of movement is the most important part of the treatment. We call it facilitation because we get the child to move spontaneously in response to special techniques of handling.

The first movement patterns in normal infants are responses to being handled during the first 6 months of life. For a long time the normal baby cannot do much for himself or get around from one place to another. He is at first totally dependent on being handled, picked up, put down, washed, fed, bathed, dressed, undressed, carried, sat up, etc. However, he is not passive during these procedures. He is practising and developing all the movements which he will later do actively, i.e. automatic protective movement patterns like holding his head, adjusting his arms and legs to normal positions when being left unsupported or left in an uncomfortable position, and holding on when being carried. As he becomes less helpless, he helps while he is dressed or washed, and fed, or he may resist volun-

tarily if he does not like it. All the time he learns movement patterns which he will need when he does the same things actively and by himself later on. These are the reactions we try to facilitate in the cerebral palsied children, who are quite helpless and may remain so. As you will see, our whole treatment is aimed at getting normal motor responses to handling.'

ORTHOPAEDIC MEASURES AND THE NEUROLOGICAL TREATMENT OF CEREBRAL PALSY

Following special requests, Dr. Bobath agreed to outline what he felt was the place of orthopaedic measures in the neurological treatment of cerebral palsy. He quoted from a current standard book on the treatment of cerebral palsy which stated that:

'Orthopaedic surgery and bracing have a limited application in cerebral palsy, being useful primarily in cases of spastic paralysis. Even in these cases they are incomplete forms of therapy without physical medicine. They are adjuncts to physical therapy. Orthopaedic surgery is used to relieve barriers of function, such as are caused by fixed deformity. It has been found more useful in the lower extremity when stability is the object.'

With this statement we feel there can be no quarrel. The approach to treatment along neurological lines can do a great deal to prevent the occurrence of deformities by the early recognition and treatment of this condition and in this way reduce the necessity for subsequent surgical measures. If deformities have already developed, a period of preliminary treatment can reduce spasticity and create most favourable conditions of surgery. Skilled after-treatment can contribute to the success of any operation. It must be realized that, fundamentally, cerebral palsy is a neurological condition and that orthopaedic problems only arise after a while if the child is encouraged to use his abnormal functional patterns over a period of time, and is allowed to remain fixed in abnormal postures. The young baby with cerebral palsy has no deformities.

There are two ways of improving a child's control over his muscle action—two approaches to treatment, orthopaedic or neurological.

In the *orthopaedic approach* the patient's state of spasticity or athetosis is taken for granted. The aim is to get him on his feet to stand and walk and to use his hands as soon as possible for all tasks that are in keeping with his chronological age. The manner of his walking and using his hands is of secondary importance to getting the functional abilities going. The patient is encouraged to use 'what he has got,' i.e. the lesser or non-affected parts for compensation (e.g. arms and trunk for

walking) and abnormal patterns of arms and hands for manual skills.

Orthopaedic measures applied to legs will only help a patient to walk if his head and trunk control is good, and if he can use his arms and hands to manipulate crutches or sticks or hold on for balance and support.

Long braces with or without a pelvic band will control the legs in standing and walking in a static position of extended hips and knees with dorsiflexed feet. When unlocked at the knees the braces will hold the legs in 90° flexion at the ankles and knees, but the pelvic band will prevent full flexion at the hips in sitting, so that the child has to compensate with a kyphosis of the spine in order to keep the trunk forward. Some degrees of abduction might be achieved by such bracing and by using abduction splints at night, but inward rotation of the legs cannot be controlled. Surgery might be added in cases where contractures have already occurred, i.e. when bracing has been attempted too late.

Short braces are usually used in hemiplegic children and in athetoids with mobile legs who tend to stand on their toes when erect. These braces are usually ineffective because, even if they fit the child and keep the feet down, when measured in supine or sitting they will not control the strong extensor spasm in standing and walking, and though the heel of the boot may be on the ground, the heel of the foot is usually up in the boot. The strength of spasticity in standing needs much stronger control than a short leg brace can give.

Some severe athetoids are given 'control braces,' i.e. long braces to the legs to make them sit more safely and thus to give the trunk and arms a better chance for manual activities. However, in these children the legs are usually much less affected than the head, trunk and arms and they would not need bracing for the legs to avoid deformities. They do not need them to stand and walk, because they could learn to do this without bracing if they could use their hands and acquire sufficient head and trunk control.

Preventive bracing is used in many young children who have not yet acquired deformities. This is a sound procedure if one decides at an early age that the child will be unable to walk in more normal ways and without bracing.

The effects of bracing are as follows:

- i. It keeps the limbs in one static posture desirable for one function, either sitting or standing. It creates a 'deformity' in a desirable postural pattern.
- ii. It does not lower spasticity, but it redistributes muscle tone, e.g. lowers tone in plantar flexors of feet and increases it in the dorsiflexors (full plantar

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flexion is not possible after long bracing) or it reduces spasticity in the hamstrings and increases it in the quadriceps, so that flexion of the knee becomes more difficult while extension is easier. Flexion of hips and lordosis will be increased.

iii. It prevents the child from moving and therefore acquiring the normal variety of movement patterns needed for rolling, sitting, kneeling, crawling, getting up and for walking. The child drags his braced legs by using trunk and hands.

iv. Muscle wasting, fibrosis of muscles, circulatory deficiency and physiological ankylosis of joints are unavoidable. This cannot be counteracted by even daily physiotherapy.

v. Bracing can only be discontinued with success (and that is without the patient losing the effect of it) after those changes in muscles and joints have become permanent. If bracing is discontinued too soon, the child returns to its former state.

vi. It prevents normal sensory experience in the muscles and joints of a limb which is confined in splints.

In the *neurological treatment* the aim is to suppress pathological reactions, producing more normal tone throughout the body musculature, not a redistribution of spasticity. The aim is also to develop and facilitate normal patterns in proper sequence or to fill in gaps—to get more normal function of higher patterns. (Prepare one activity by another.)

This cannot be combined with the orthopaedic approach because they would cancel each other out. We want to change patients' motor patterns in order to obtain a greater variety of more normal quality. After long-standing bracing, changes of patterns are impossible. Fibrosis of muscles, ankylosis of joints make introduction of different patterns impossible. Thus control by special furniture and bracing from outside deprives the child of using whatever we get going of his own control in treatment.

We also want to give the child more normal sensori-motor patterns. For this the child has to be mobile—to do his movements with normal effort and feel the normal sensations of the weight of his limbs (without the weight and pressure of braces and appliances).

There cannot be a carry-over of our treatment if, in between treatments, the child is fixed in, and loaded with braces.

SURGERY AND TEMPORARY PLASTERS

This might be necessary and can be considered with neurological treatment where it has failed, or in older cases where structural deformities have occurred. It is an adjunct, to be used with great discretion and in properly selected cases. It should be taken into account that 'shifting' of spasticity may occur, e.g. that the straightening of one joint may be followed by changes

at a neighbouring one. Also, that by weakening one pattern, say of flexion, one may get an excess of another, e.g. extension, or *vice versa*. This is very possible in athetoids. A temporary plaster may be a means of trying out the effects on neighbouring joints before deciding on final surgery.

Surgery does not restrict the movements of the child, nor does it produce such static changes as long-standing bracing does.

THE LINK-UP OF THE DIFFERENT ASPECTS OF TREATMENT

This was the final paper, read by Mrs. Bobath. She stated that cerebral palsy was a condition affecting the whole child and though he may have difficulties in various fields of activity, e.g. movement, speech, hearing, vision, mental development, sensory perception, emotional stability and social adjustment, all these were related to one other. Because of this, the various aspects of treatment, and the child's management at home and at school, should be linked up, and each therapist working in her specialized field should at all times treat the whole child.

SPEECH THERAPY

Many of these children have speech and hearing defects, especially when the upper extremities and the neck are affected. As we are dealing with one person, with one central nervous system, speech therapy cannot be separated from the patient's physical treatment and, therefore, the close cooperation of the physiotherapist and the speech therapist is of great importance in treatment.

Just as the physiotherapist aims to give normal sensations of movement and muscle tone, so does the speech therapist aim at giving normal sensations of speech. With the physiotherapist taking the lead, breaking up and stopping abnormal reactions of the body, the speech therapist continues to break up and prevent abnormal reactions in the speech musculature and of the body, associated with speech. Not until a patient has a certain amount of head control, can he hope to gain control over the finer movements necessary for speech. Therefore a patient receives physiotherapy for some time before he receives formal speech therapy; but advice to parents from the speech therapist with regard to feeding, swallowing, biting, chewing and babbling is given. All these functions play an important role in the patient's general language and speech development.

The speech therapist must know how to position and move the child to keep muscle tone normal generally and to prevent associated reactions, i.e. speech independent of body movements. Then she goes through all the stages of normal speech development, from breathing, voicing, babbling and finally to words and phrases. The patient is given the chance to feel and hear the normal sounds without associated abnormal movements. This is all done under conditions which have some relation to real life. Just as life is stimulating, so is treatment, and gradually the patient learns to adjust himself normally to it.

We are treating the whole child, and therefore we need an understanding of his emotional and social needs, and knowledge of the home and school background and the relation of the problems found there to treatment is essential for the success of treatment.

OCCUPATIONAL THERAPY

The occupational therapist also should be aware of the fact that the use of the hands for skilled activities depends on the total motor behaviour of the child. Even in the normal child the independent use of the hands is achieved only at 18 months, i.e. at a time when the child does not need his hands any longer for support, when his balance in sitting, standing and walking has been established. There are many factors that have to be taken into account, of which I only shall mention a few. For instance:

(a) The importance of developing head and trunk control to ensure independent movements of the arm into all directions.

(b) The influence of asymmetrical tonic neck reflexes on the arms: using both hands together, eye-hand control, getting the hand to the face or into midline towards the body.

(c) The influence of total flexor patterns in sitting.

(d) The influence of the total flexor pattern on extension of the arms, wrist and fingers, on abduction of fingers and thumb, opposition, supination, etc. Most activities like dressing and feeding need the palms turned towards the body.

All this shows that the use of the hands depends very much on the motor behaviour of the body. The occupational therapist will find that certain movements of the arms and hands are easier with the child in certain positions. Because of this we need very close cooperation of physio- and occupational therapists.

However, the physiotherapist, the speech therapist and the occupational therapist see the child only for very short times, even if the child is treated daily. The child spends most of his life at home and at school, and if we

want a good carry-over of the treatment we have to get the parents and the teachers to understand the problems of the child, and to keep them constantly informed of the child's abilities and difficulties at the various stages of treatment. They have to know what we are doing with the child and to see how the child reacts to it. This means that they have to be present to see the treatment at intervals, so that we can show them how to handle the child in order to make their movements easier in every-day living and learning.

The first thing in this respect is to teach the parents and teachers how to observe the children—how to distinguish the abnormal behaviour patterns from the normal ones. We have also to explain, to some extent, the sequence in which normal motor patterns develop, so that they know what to expect first, second and later on. Some parents, after first worrying when their baby appears to be different from a normal child, tend to accept as inevitable his abnormal postures and difficulties or his inability to move. Thus one often finds a child of 2 years of age being treated like a baby of 4 weeks. The child becomes spoiled because everything is done for him. By assuming he is incapable of normal development, the parents unwittingly aggravate the difficulties.

This brings us to the second point, viz. that we have to teach the parents and the teachers how to help the child gradually to take over more and more normal activities. They must be shown that the child is less spastic in certain positions, that certain parts of a movement may produce spasticity, while other parts of the same movement can be done fairly normally. They should learn to recognize the stage of a movement at which help should be given to avoid spasticity, and the stage at which the child should be allowed to take over without assistance. For instance, the child should be allowed to assist in dressing and undressing with whatever movements he does fairly normally, but should be helped in between, when necessary. Too much, however, would not be expected of the child in the first place, and to avoid this it is necessary to know which are the easier and more primitive movements and which are the later and more difficult ones.

For example, take *dressing*: The normal child cooperates in dressing in a definite sequence. At about one year of age he will put out his foot to have his shoes put on; but he will be about 2 years at least before he can put them on by himself;

Take *feeding* for another example: Here the beginnings of the early pattern of self-feeding show themselves when the child is about 4 or 5 weeks old—when he puts the fingers of one hand in his mouth. At about 24 weeks he can bring both hands to his mouth, and he starts to suck them. Each simple accomplishment thus gradually contributes to the stage, reached at about 36 weeks, when he is able to grasp an object and take it to his mouth. So a child with cerebral palsy cannot, even at 3 or 4 years of age, be expected to feed himself with a spoon, before he is able to get his hand to his mouth.

The third point is to convey to the parents and teachers the need to encourage the child's own initiative—to prepare the way for independent action in as normal a manner as possible. The abilities of the child expand with progress in treatment and, therefore, his everyday activities should expand correspondingly. Parents and teachers should be kept informed continuously about the child's readiness for more advanced activities, otherwise they may continue to help him when it is no longer necessary. On the other hand, they should be advised not to allow the child to do things in grossly abnormal ways.

To sum up, we have:

1. To teach the parents and teachers to observe and recognize the abnormal behaviour patterns of the children;

2. To teach them how to help the children to take over more and more normal activities; and

3. To teach them to make the child as independent as possible within his own capabilities at various stages of treatment.

The problem of the older child is a much more difficult one. Psychologically it would be a bad thing for the older child with established faulty habits of sitting, feeding, dressing, etc. to stop these activities entirely. We must not give the child the feeling that he has to abandon an achievement of which he may be very proud. On the other hand, if his movements are too abnormal, one can always stop him from doing too much. For instance, if he walks very badly one can avoid taking him for long walks and give him a tricycle instead. Furthermore, as the child improves under treatment, all his everyday movements will become easier and more normal. Even without being taught, the child may suddenly do something that he has never done before. A young man of 28 years who never could clean himself after his toilet, suddenly did it for the first time in his life after having achieved better balance in standing.

It is amazing how soon the parents get used to the child's abnormal postures and

movements, and also how they accept the idea that the child cannot do certain things. They will forever go on helping and not give the child a chance. One must not overlook the fact that the one compensation for having a handicapped child is that the mother has a child whom she can regard as a perpetual baby. Therefore, whenever the child is ready to help himself, we should see that he is given an incentive to do so. Anything the child does fairly normally should be encouraged, because it will make him better; but anything he does grossly abnormally should not be allowed because it will make him worse, that is to say, more spastic or more athetoid.

The child must be given as much confidence and independence as his capacity allows, both physically and mentally. He should be praised for every little achievement. In this way he will develop a sense of security and independence. Constant nagging and reminding the child, e.g. to hold his head up or to straighten his back, will give him a sense of failure and insecurity. Instead of this, one can without saying a word put the child's head into a correct position, straighten his back, push a hand or a foot in place, etc. This sort of reminder of what he should do is much more effective and easier for the child to accept than talking to him.

You have seen how we help the children in treatment to change their abnormal postures and movements to more normal ones. To carry over this work at home and at school we show the parents and teachers how to make use of certain postures which make movements easier, and how to avoid others that increase spasticity and limit movements. The mother is always with us during treatment, the teacher sometimes. In day-schools and in residential centres the physical therapist also sees the children during school hours whenever the teacher needs advice on the child's physical management.

The parent and the teacher should realize that no one or two postures, even if corrected, should be maintained for long periods of time. One only has to remember that the normal child is hardly ever still, and even an adult person when sitting constantly changes his posture to avoid getting stiff. The child who has gained some sitting balance in treatment should learn to sit on various types of chairs (or stools, if possible), and he also should be able to sit in various positions on the floor. Tables should be adjusted in height so that the child with a tendency to flexion will sit on a low chair with a high table, and a child with

a tendency to extension on a high chair with a low table. The child should be able to move his head, shoulders and legs freely and only be given the absolute minimum of support from outside by adjusting ordinary chairs to the needs of the individual child. In this way, and with the necessary preparation in physical therapy, the child learns to control his posture and gets his balance. If the child, who is not yet able to sit alone, sits for long periods of the day in a special chair, in one corrected posture with straight back, and flexion at hips, knees and ankles, he is in danger of developing contractures of the hips and knees; and when he is made to sit with extended knees his hips will not be properly flexed and he will sit back on his sacrum and develop a kyphosis of the spine in his attempts to maintain the sitting posture.

All children with cerebral palsy, especially those who have been made to sit, tend to have too much flexion of the trunk and hips, which makes upright standing and walking difficult or impossible. For this reason, and to counteract such tendencies, all children (regardless of their age) should spend some considerable time of the day lying prone; and this position, made more comfortable with a small pillow under the chest, can be used for school work in preference to sitting. Books can be placed on book-stands to make the child look forward and up rather than downward, as they usually do. Book-stands are also useful on tables when sitting in a chair, and reading in this way counteracts the strong tendency to flexion of the trunk and arms. Writing and drawing can be done well in prone lying, supported on the forearms, and sometimes gives better results than when done sitting, because of the stabilizing effect of this position on the shoulder girdle.

Young children who cannot sit properly, i.e. with correct balance and unsupported, will be better sitting on the floor than on chairs. Normal babies do not start sitting on a chair either; they first sit in their cots or prams, or on the floor. But before they sit at all, they develop their back muscles by lying on their tummies. The young child with cerebral palsy who sits unsteadily and with a round back should spend a lot of his play-time on his tummy, rolling around and moving freely on the floor, playing with toys, looking at books or television. When the child has learned to crawl he can spend some of his play-time in upright kneeling, playing with toys on a low table. This enables him to get extension and control of balance at the hips in preparation

for standing and walking. When the child stands up and walks we should see that his balance does not entirely depend on grasping and holding on with his hands. We train the children to balance with their head, trunk, hips and legs. They should rather support themselves with their extended hands on furniture or against walls, than by holding on to bars or rails, as this will cause too much flexion not only in the arms and hands, but also throughout the whole body.

When the child is able to stand fairly well in treatment, he should be encouraged to play in this position at home and at school as well; but care should be taken when he is standing at, say, a table, that his heels are on the floor.

When the child starts to walk, he should do so only for short periods of time at first. We should give him some support as long as this makes his walking more normal, even if only by one finger to help him to balance. The point at which help is given, of course, varies with every child. We show the parents and teachers not only the correct points at which to hold the child, but also what will go wrong if the support is given in the wrong places.

I hope I have given you an idea of the principles of our treatment and general management of the child's physical condition. But physical, mental and emotional development are not separate entities. They depend on one another, they influence one another, and with physical improvement goes usually social adjustment, emotional stability and even improvement of the child's mentality in many cases.

The sensori-motor problem of the child is the one that embraces all other problems, which include the emotional, mental and social difficulties. Too often these various aspects of one total behaviour problem are divided up, and treated separately without relating them to one another. Too often the importance of each one is weighed up against that of the others; and too often the motor habilitation is said to be the least important as compared with social and emotional adjustment and mental ability. The sensory aspect is often stressed, but again unrelated to any of the other problems. Sensori-motor development, however, is the basis for all our experiences, for all our learning, for our ability to adjust ourselves to the environment, to the people we live with and to the objects we have to handle. If we want to treat the whole child we have to start from there, and we should branch out from there when we want to give special treatment and attention to other associated handicaps.

Mr. J. A. Pierre are no Centre; Johann Dr. consult 50, (Telep

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NOTES AND NEWS : BERIGTE

Mr. Jack Penn, F.R.C.S., of Johannesburg and Mr. Pierre Hugo, M.Ch., of Pretoria, Plastic Surgeons, are now in practice in partnership at Clarendon Centre, Clarendon Place, 4 Park Lane, Parktown, Johannesburg. (Telephone: 44-9587).

Dr. Hugo will also continue to be available for consultation by appointment at his old address:

50, Van Riebeeck Medical Building, Pretoria. (Telephone: 30764).

* * *

Mr. Leon Bryer, M.Ch.(Ortho.), F.R.C.S. (Ed.), has commenced practice as an Orthopaedic Surgeon at 202 Osler Chambers, corner of Jeppe and Delvers Streets, Johannesburg. (Telephones: Rooms: 22-8703; Residence: 44-1648).

* * *

Dr. L. J. du Preez (formerly of St. Thomas' Hospital, London; American University, Beirut; Massachusetts General Hospital and Harvard University, Boston) has joined Drs. Loots, Osler, Esterhuizen and Hurwitz in their radiological practice at 101 Medical Centre, 209 Jeppe Street, Johannesburg.

* * *

Dr. Maurice H. Luntz, M.B., Ch.B. (U.C.T.), D.O.M.S. (Dub.), D.O. (Lond.), F.R.C.S. (Ophth.), recently of St. Mary's Hospital and Medical School, London, has commenced practice as an ophthalmic surgeon in partnership with Drs. O. M. Haaburger and Charles Goldin at 303 Dumbarton House, Church Street, Cape Town. (Telephones: Rooms: 2-6106; Residence: 77-1225).

* * *

ASSOCIATION OF PHYSICIANS OF SOUTH AFRICA

SOUTHERN TRANSVAAL GROUP

Arrangements are nearing completion for what promises to be a highly interesting Scientific Meeting in Johannesburg, from 6 to 9 July 1960. Forty papers on a wide variety of subjects are being presented.

The programme includes a session at the Pneumococcal Bureau, where papers on respiratory physiology and the measurement of pulmonary function will be read.

Under the auspices of the Endocrine Society, a session will be devoted to *Diabetes and Thyroid Disease*.

On Saturday morning 9 July there will be a clinical meeting at Baragwanath Hospital with special reference to problems in African cardiology.

PREPARATIONS AND APPLIANCES

DISAMIDE FOR THE CONTROL OF OEDEMA

British Drug Houses announce the introduction of *Disamide*, a product of original research for use in the control of oedema due to a variety of conditions.

Composition: *Disamide* is 5-chloro-2:4-disulphamyltoluene a substance first synthesized and studied in the B.D.H. Research Laboratories. *Disamide* is not a 'sulphonamide' and does not there-

fore share the toxic properties of the antibacterial sulphonamides which are substituted anilines, i.e. substituted p-aminobenzene derivatives.

Action: When taken orally, *Disamide* exerts a diuretic action which reaches a maximum 2 hours after administration. It also causes marked increase in the elimination of sodium and chloride ions.

Indications: *Disamide* is indicated for oedema associated with congestive heart failure, nephrotic

Instead of the usual Dinner, there will be a Sun-downer and Snack Party from 6-8 p.m. on Friday, 8 July. A list of plays and other entertainments which will be on in Johannesburg at the time will be circulated early in June and bookings for these will gladly be arranged. Furthermore, our Committee will be pleased to reserve hotel accommodation for visitors.

Further information may be obtained from the Honorary Organizing Secretary, 504 Medical Centre, Jeppe Street, Johannesburg. (Telephone: 22-3837).

* * *

WESTDENE PRODUCTS SCHOLARSHIPS

The following awards have been made to 4th, 5th and 6th year medical students at the undermentioned Universities. In the case of Stellenbosch University awards were only made to 4th and 5th year students as, at present, no provision is made for 6th year medical students; 6th year students will be catered for in 1961. Each Westdene Products Scholarship is worth £100.

The awards are made to medical students of high academic standards and showing a sense of social responsibility by taking an active part in student and other affairs.

To date 96 awards have been made.

In addition to these medical scholarships there is the Westdene Products Nursing Scholarship for Industrial and Scientific Investigation and the Westdene Products Scholarship for Pharmaceutical Study, Research and Development of £200 each, yearly.

Cape Town University

4th year: P. J. van Boxel.

5th year: S. Gordon.

6th year: W. Gevers.

Stellenbosch University

4th year: Miss C. Malan.

5th year: R. Schoonees.

Natal University

4th year: O. M. Jolobe.

5th year: J. R. Domingo.

6th year: D. B. Mathloko.

Pretoria University

4th year: O. W. Prozesky.

5th year: P. J. Schutte.

6th year: J. M. Hugo.

Witwatersrand University

4th year: Clive Rosendorff.

5th year: Miss Judith Issroff.

6th year: Arthur H. Rubenstein.

syndrome, toxæmia of pregnancy, cirrhosis of the liver, steroid-induced oedema, chronic glomerulonephritis, obesity with fluid retention and oedema of premenstrual tension.

Precautions: When a patient is being maintained on *Disamide* for a long time it is advisable to administer potassium supplements by mouth. If potassium supplementation is required, it is preferable that it should be carried out on those days on which *Disamide* is not taken. The required daily dose of potassium chloride on these occasions is up to 4 g. Patients taking *Disamide* Tablets do not need to be maintained on a strict salt-free diet which may lead to increased potassium loss, and are probably best kept on an ordinary diet with no added salt.

Dosage (Average): Two tablets for 5 days each week reducing to 1-2 tablets on alternate days to avoid further fluid retention.

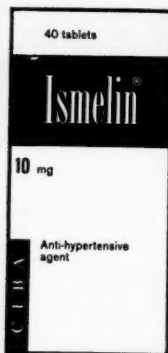
Mode of Issue: Tablets (scored) each containing 100 mg. of 5-chloro-2-4:disulphamyltoluene.

Bottles of 25, 100 and 500 tablets.

ISMELIN FOR HYPERTENSION

A NEW APPROACH TO ANTI-HYPERTENSIVE TREATMENT

Ismelin (guanethidine), developed in the CIBA Research Laboratories, is an anti-hypertensive substance which possesses a new-type mechanism of action and whose hypotensive effect is more potent than that of any other anti-hypertensive now in common use.



Pharmacological analysis and clinical trials have shown that the mode of action of *Ismelin* is different from that of other anti-hypertensives. It is, in fact, a new type of drug, to which the term 'sympathetic nervous inhibitor' has been applied.

Ismelin is indicated in all forms of hypertension, including especially cases where the disease has reached an advanced stage and is refractory to other anti-hypertensive agents.

Thanks to its highly potent effect, *Ismelin* is capable of reducing the blood pressure in some 90% of cases of hypertension. Since *Ismelin* is an extremely active drug, the dosage must be individualized;

treatment should in principle be initiated with small doses, which are then gradually raised, depending on the patient's response.

Packages: 10 mg. tablets, 40's. 25 mg. tablets, 30's.

Further Information from: Ciba (Pty.) Ltd., P.O. Box 5383, Johannesburg. (Telephone: 33-7491).

THE RETIX UNIT FOR COMPLETE DIATHERMY

Westdene Products (Pty.) Ltd. have pleasure in introducing the new *Retix Unit* (manufactured by R. Toury, France). The *Retix Unit* is the complete diathermy apparatus for ophthalmologists, and can be used in operating rooms as well as in doctors' offices, where it has multiple uses.

Specially designed for detachment of the retina operation, with coagulations of up to 50 milliamperes for retinal detachment with perforating electrodes, and coagulation up to 200 milliamperes with surface electrodes.

Cutting and coagulation circuit.

Ophthalmological diathermy treatment up to 1 ampere.

Variable low tension light circuit for diagnostic instruments from 0 to 12 volts.

Special circuit for general cautery.

The 50 and 200 milliamperes circuits for electrocoagulations of detachments of the retina have a special adjustment circuit and device for intensity regulation, which assures regular coagulations without any risk of accident by over-intensity.

The spark gap with single adjustment and luminous control is removable and assures long-lasting stability.

R. Toury, France, are manufacturers of Cautery Machines, Examination



Lamps, Electro-Surgical Units for use in general surgery, neuro-surgery, pulmonary surgery and prostatic resection, suction units, ultra-violet lamps and infra-red lamps of the finest quality.

All enquiries should be addressed to:

Westdene Products (Pty.) Ltd., 23 Essanby House, 175 Jeppe Street, Johannesburg, or to their Branches in Cape Town, Durban and Pretoria.

CORRESPONDENCE

CYTOSTATIC PREPARATION—CYTOXAN: ENDOXAN

To the Editor: Recently a SAPA-Associated Press cable from U.S.A. concerning new cytostatic preparations based on nitrogen mustard has been given considerable publicity in the lay press of this country, and special reference has been made to a promising preparation *Cytosoxan* (brand); see e.g. the *Pretoria News* of 12 April 1960, page 6: 'Gas is Ally in Bid to Beat Cancer.'

To avoid confusion amongst medical practitioners in South Africa, we wish to advise that this preparation is also available to the medical profession here under its original name *Endoxan*. *Cytosoxan* is the

brand name used in the United States for the same product.

Endoxan: *Cytosoxan* has been developed in the research laboratories of a German pharmaceutical house, Asta-Werke A.G., Brackwede. Clinical tests are also being conducted in teaching hospitals of this country, and a first report has been published in the 27 February 1960 issue of *Geneeskunde*, Vol. 2, No. 2, pp. 40-47.

H. M. Snyckers,
Director.

Noristan Laboratories (Pty.) Limited,
Silverton, Pretoria.